Dissertation on

SERUM LIPID PROFILE AND URIC ACID CHANGES DUE TO PASSIVE SMOKING IN HEALTHY WOMEN

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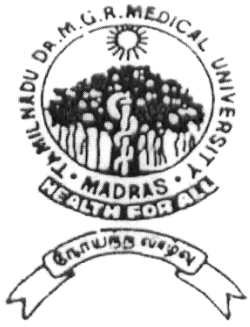

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON SERUM LIPID PROFILE AND URIC ACID CHANGES DUE TO PASSIVE SMOKING IN HEALTHY WOMEN"** submitted by **Dr.SURENDRAMOHAN.T,** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2008 is a bona fide record of work done by him under my direct audience and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON SERUM LIPID PROFILE AND URIC ACID CHANGES DUE TO PASSIVE SMOKING IN HEALTHY WOMEN"** is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2005-2008 under the guidance and supervision of **Prof. P. Thirumalaikolundu Subramanian M.D.**

The dissertation is submitted to The Tamilnadu **Dr.M.G.R.Medical University** towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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CONTENTS

INTRODUCTION

There have been few epidemics as devastating and preventable as that caused by tobacco consumption. Cigarette smoking became highly prevalent through the 20th century; with a lag of several decades, the rise of smoking was followed by epidemic increases in the diseases now known to be caused by smoking, including lung and other cancers, heart disease, and chronic lung disease. By mid-century, epidemiologic studies were providing the initial evidence establishing that smoking caused these diseases. As the 21st century begins, smoking is on the rise in developing countries, even as it has started declining in the developed countries.

Some of the first epidemiological studies on secondhand smoke and health were reported in the late 1960s [1-3]. One German physician, Fritz Lickint, used the term "passive smoking" in his 1939 book on smoking [4]. The 2006 Surgeon General's report uses the term "secondhand smoke," although the term "environmental tobacco smoke" (ETS) was used more frequently in earlier reports [5].

The first major studies on secondhand smoke and lung cancer in nonsmokers were reported in 1981[8,9], and by 1986 the evidence supported the conclusion that secondhand smoke was a cause of lung cancer in non-smokers, a conclusion reached by the International Agency for Research on Cancer (IARC)[7,10,11]. Subsequently, a now substantial body of evidence has continued to identify new diseases and other adverse effects of secondhand smoke, including increased risk for coronary heart disease [6,12-15].

The tobacco industry's campaign to discredit the evidence on active smoking initially and on secondhand smoke subsequently is now well documented as legal actions against the tobacco industry. There was an attempt to maintain seeming controversy among scientists even as the evidence mounted and supported converging conclusions by expert panels on the adverse effects of secondhand smoke.

Environmental tobacco smoke (ETS) is a combination of side stream smoke and exhaled mainstream smoke, while passive smoking is defined as an involuntary exposure to ETS [16]. Little attention has been paid in the past to health consequences of ETS. There has been a growing concern that non-smokers exposed at

home or at work place may also face increased risks of diseases similar to those described in smokers [16].

Exposure to ETS reduces the blood's ability to carry oxygen to the heart and compromises the ability of the myocardium to produce ATP using oxygen. These effects manifest as reduced exercise capacity in people exposed to ETS. Passive smoking over a period of ten years changes the intima /media ratio by enhancing the thickness of the vessel wall and by adversely affecting endothelial function. ETS adversely affects platelet function, depresses cellular respiration at the level of the mitochondria. It also potentates other risk factors like hypertension [17].

It is well known that cigarette smoking is associated with elevation of plasma lipids and changes in lipoprotein distribution [18]. Smokers have elevated serum uric acid levels and that these levels denote the level of oxidative stress imparted to the body by smoking [67]. Although the effect of passive smoking on human health has been established as a major risk factor, the effect of passive smoking on lipid profile and serum uric acid has not been well established. The present study is aimed at correlating the lipid profile and the serum uric acid in passive smokers and to prove their atherogenic potential.

AIM AND OBJECTIVES

- To study the effects of passive smoking on lipid profile and to estimate serum uric acid among healthy women whose husbands are smokers.
- To correlate the biochemical changes with the duration of exposure.
- To assess the atherogenic potential of environmental tobacco smoke.

REVIEW OF LITERATURE

The ancient Indians not only enjoyed its stimulatory effects of tobacco but also believed it has medicinal properties. Until the late 1880s, cigarette smoking was less frequent than other forms of tobacco usage, e.g., snuff. But the invention in 1880s of cigarette rolling machine followed by the development of safety matches tilted the balance in favor of cigarette smoking [19].

Tobacco smoke contains over 4000 different chemicals, many of which have adverse effects on human health and can contribute to the development of diseases such as heart disease, stroke and lung cancer. Cigarette smoking is addictive, because of the presence of the alkaloid nicotine and withdrawal of this causes the unpleasant side-effects of quitting. Cigarette smoking is also associated with a large number of physiological changes particularly in blood lipids and hemostatic factors that explain its role in coronary heart disease [20, 21].

ENVIRONMENTAL TOBACCO SMOKE (ETS) (SECONDHAND SMOKE)

The process of smoking produces three different types of tobacco smoke:

(i) Mainstream smoke: the smoke directly inhaled through the burning cigarette by the smoker [22].

(ii) Exhaled mainstream smoke: The smoke breathed out by the smokers from their lungs. The composition of the mainstream and the exhaled mainstream smoke is likely to differ, some of the compounds in smoke being retained by the smoker or otherwise altered by the process [22].

(iii) Sidestream smoke: The smoke which drifts away from the burning end of the lit cigarette [22] .

Mainstream and sidestream smoke consist of a large number of chemical carcinogens and other toxic substances, but undiluted sidestream smoke contains many compounds in far greater concentration. For example, side stream smoke contains greater amounts of ammonia, benzene, carbon monoxide, nicotine and the carcinogens 2-napthylamine, 4-aminobiphenyl, N-nitrosamine,

benzanthracene and benzopyrene per milligram of tobacco burned [23].

The particles of sidestream smoke are smaller than those of mainstream smoke, meaning that they may be inhaled deeply into the lungs and is considered by the Environment Protection Agency to be in the same class as radon and asbestos [23].

Although it has been considered that 85% of the smoke present in an average room in which smoking has occurred is composed of sidestream smoke, passive smokers still have a lower exposure to the harmful components of tobacco smoke than active smokers, who draw smoke directly into their lungs. Though the amount of smoke inhaled by smokers is only a fraction of the amount inhaled by smokers, a wealth of scientific evidence now exists showing that the breathing of tobacco smoke polluted by non-smokers can lead to serious harm [22].

Sidestream smoke has been found to contain virtually all the carcinogens identified in the smoke directly by smokers. Since side stream smoke does not pass through the filters of the cigarettes; it contains around 100 times the weight of the carcinogens of mainstream smoke. In an autopsy study of

nonsmoking women who had been married to smokers, significantly more epithelial, possibly precancerous lesions were found in the bronchi and pulmonary parenchyma than in wives of non-smokers. The risk of lung cancer was increased by approximately 20 percent for non-smoking women married to smokers.

ESTABLISHED AND POTENTIAL HEALTH EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE [24]

Established

- Increased lower respiratory infections in children
- Increased respiratory symptoms in children
- Reduced lung growth in children
- Increased lung cancer in non-smokers
- Irritation of the eyes, nose, throat and lower respiratory tract
- Increased risk of cardiovascular disease

Potential

- Increased respiratory symptoms in adults
- Reduced lung function in adults
- Exacerbation of asthma

- Increased risk for non-respiratory cancers
- Increased risk of sudden infant death
- Reduced birth-weight
- Increased risk of stroke

Effects in children

Exposure to ETS has been found to be a cause of slightly reduced birth weight, lower respiratory illnesses, chronic respiratory symptoms, middle ear disease, reduced lung function, asthma among children of school age and maternal smoking has been characterized as a major cause of sudden infant death syndrome (SIDS).

Growth and development — Active smoking by pregnant women, resulting in secondhand smoke (SHS) exposure for the developing fetus, increases risk for a variety of adverse health effects in children including reduced birth weight as well, estimates of the reduction of birth weight associated with paternal smoking ranging from 24 to 31 grams [25,26]. Adverse effects of in utero or postnatal exposure to SHS on neuropsychological development and physical growth have also been postulated. Other

nonfatal effects possibly are growth retardation and congenital malformations [27-29].

Sudden infant death syndrome — Sudden infant death syndrome (SIDS) refers to the unexpected death of a seemingly healthy infant while asleep. Studies measured maternal smoking after pregnancy, along with paternal smoking and household smoking generally. One report goes on to state that tobacco smoke exposure is one of the major preventable risk factors for SIDS [14, 15].

Lower respiratory tract illnesses — Infants with smoking parents have a significantly increased frequency of bronchitis and pneumonia during the first year of life [5, 30-33]. Presumably this association represents an increase in frequency or severity of illnesses that are infectious in etiology and not a direct response of the lung to the toxic components of SHS. An effect has not been readily identified after the first few years of life; this finding may be explained by higher exposures because of the time-activity patterns of young infants, which place them in close proximity to cigarettes smoked by their mothers.

Respiratory symptoms and illness —a greater frequency of the most common respiratory symptoms: cough, phlegm, and wheeze, in the children of smokers is seen. Having both parents smoke was associated with the highest levels of risk [7, 12, 15, 34]

Asthma —is a long-term consequence of the increased occurrence of lower respiratory infection in early childhood, or through other pathophysiological mechanisms including inflammation of the respiratory epithelium [5, 35, 36]. In utero exposures from maternal smoking may also affect lung development and increase the risk for asthma.. Maternal smoking during pregnancy also reduced ventilatory function measured shortly after birth. Exposure to secondhand smoke during childhood is also associated with increased prevalence of asthma in adults [37]. Guidelines for the management of asthma all urge reduction of SHS exposure at home [38].

Lung growth and development — during childhood, measures of lung function increase are more or less parallel to increase in height. In a study of 193 high school athletes, for example, there was a fourfold increase in incidence of low forced expiratory flow 25 to 75 percent (FEF25-75) and/or cough in

athletes exposed to secondhand smoke compared with athletes not exposed [39].

Middle ear disease —Exposure to SHS has been most consistently associated with recurrent otitis media and not with incident or single episodes. The evidence to date supports a causal relationship [5, 14].

Dental caries — Exposure to SHS may be associated with an increased risk of dental caries in children. Studies found that elevated cotinine levels were associated with caries in deciduous but not permanent teeth [40].

Effects in adults

Lung cancer — the exposure to carcinogens with SHS is far less than the exposure that occurs with active smoking. On the other hand, exposure to SHS can begin in childhood and extend across the full lifespan. Genotoxic activity, the ability to damage DNA, has been demonstrated for many components of SHS [41-43]. A number of studies have shown that SHS is associated with lung cancer. There appears to be a dose-response relationship between intensity of exposure and relative risk [5-10, 44, 45].

In a prospective cohort study of 91,540 nonsmoking women in Japan, standardized mortality ratios (SMRs) for lung cancer increased significantly with the amount smoked by the husbands [6]. The findings could not be explained by confounding factors and were unchanged when follow-up of the study group was extended [44]. In a population based, case-control study, household exposure to 25 or more smoker-years during childhood and adolescence doubled the risk of lung cancer, whereas exposure to fewer than 25 smoker-years did not increase the risk [46]. In another similarly designed study, tobacco use by the spouse was associated with a 30 percent increase in risk of lung cancer. The risk rose with increasing levels of pack-year exposure from the spouse; 80 or more pack-years of exposure was associated with an 80 percent excess risk of lung cancer. A meta-analysis of 37 published studies involving 4626 people with lung cancer found an excess risk of lung cancer of 24 percent (95% CI 13-36 percent) if an individual lived with a smoker [48]. Based upon the available data, the United Kingdom's Scientific Committee on Tobacco and Health concluded that exposure to secondhand smoke is a cause of lung cancer [13]. The US Environmental Protection Agency has classified SHS as a Group A carcinogen, that is, a known human carcinogen [20].

The risk for the development of lung cancer in response to secondhand smoke may be influenced by genetics. One study found a significant increase in polymorphisms in the gene glutathione S-transferase M1 which is believed to play a role in detoxifying carcinogens in tobacco smoke; thus, mutations which decrease its activity could serve to promote tumorigenesis [50].

Cardiovascular disease — the risk of CHD in active smokers increases with amount and duration of cigarette smoking and decreases quickly with cessation. Active cigarette smoking is considered to [51] increase the risk of cardiovascular disease by promoting atherosclerosis, increase the tendency to thrombosis, cause coronary artery spasm, increase the likelihood of cardiac arrhythmias, decrease the oxygen-carrying capacity of the blood, affect vascular endothelial cell function.

It is biologically plausible that secondhand smoke could be associated with increased risk for CHD through the same mechanisms considered relevant for active smoking. One study found that 30 minutes of exposure to secondhand smoke in healthy young volunteers compromised coronary artery endothelial function in a manner that was indistinguishable from that of habitual smokers, suggesting that endothelial dysfunction may be

an important mechanism by which secondhand smoke increases CHD risk [52]. One study associated increased inflammatory markers including higher white blood cell counts and levels of C-reactive protein, homocysteine, fibrinogen, and oxidized LDL cholesterol [53] with risk of CAD. A prospective cohort study performed in 2004 measured serum cotinine levels in non-smokers exposed to SHS [54]. After 20 years of follow-up, among the 2105 men who were nonsmokers, the risk of CHD was increased in those with higher serum cotinine concentrations.

In 2005, the California Environmental Protection Agency (CalEPA), established that 22,700 to 69,000 deaths from CHD were attributable to SHS in 2000 [15]. The American Heart Association's Council on Cardiopulmonary and Critical Care concluded in 1992 that SHS both increases the risk of heart disease and is "a major preventable cause of cardiovascular disease and death" [55].

Respiratory symptoms and illnesses —Consistent evidence of an effect of passive smoking on chronic respiratory symptoms in adults has not been found.

Lung function — Exposure to secondhand smoke has been associated in cross-sectional investigations with reduction of several lung function measures. However, the findings have not been consistent [13].

Diabetes —a 15-year cohort study also found an increased incidence of glucose intolerance in young adults aged 18 to 30 exposed to SHS [56].

All-cause mortality —Nonsmoking adults who lived with smokers had about a 15 percent increase in adjusted mortality compared with those living in a smoke-free household [103]

MEASUREMENT OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Exposure to ETS, like most other environmental exposures is difficult to measure. Several methods could be used for assessment of exposure in population based research on exposure. They are:

(i) **Direct environmental measurements:**

Nicotine is a widely used marker of ETS. Tobacco products are the principle source of nicotine and beyond trace

concentrations, vapor-based nicotine is specific to tobacco and therefore suitable for measurement of ETS. Numerous field studies have been carried out on the levels of nicotine in various indoor environments and environment nicotine levels have been found to correlate with number of cigarettes smoked. In environments where smoking is permitted, components of tobacco smoke can readily be measured in the air, including small particles in the size range that penetrates into the lung, carbon monoxide, nicotine, and benzene. The exposures of nonsmokers will also depend upon proximity to smokers, particularly during the time that they are actively smoking. Concentrations of particles in homes with smokers are typically at least double those of homes without smokers. Nicotine, present as a gas in SHS, can be readily measured in indoor air using either active or passive samplers. Its presence is highly specific for tobacco smoke, as there are no other sources. Benzene, which causes leukemia, is generated by tobacco combustion and may contribute to the increased risk of leukemia in active smokers compared with nonsmokers.

(ii) Personal monitoring:

This may provide a better approximation of individual exposure than ambient monitoring. For epidemiological research,

personal monitoring of exposure to ETS has seldom been used, but it would be valuable in future research, particularly for the measurements of accumulated exposures.

(iii) Questionnaires:

Most population research into health effects of passive smoking has used questionnaires to collect information on the smoking habits of the spouse, other family members and companions in the workplace. Riboli et al (1990) showed that, when appropriately questioned, non-smoking women can provide descriptions of exposures to ETS that are consistent with urinary cotinine levels. Brownson et al (1993), Emerson et al (1995) also showed high degree of reliability in responses about of passive smoking.

(iv) Biological markers:

Secondhand smoke can also be documented by the measurement of biomarkers, that is, tobacco smoke components or metabolites of these components in body fluids of nonsmokers, including blood, urine, saliva, and also in hair. Studies using biomarkers show clearly that SHS-exposed nonsmokers absorb, metabolize, and excrete tobacco smoke components, including

nicotine and tobacco-specific carcinogens [13]. In nonsmokers, tobacco-smoke carcinogens have also been shown to be bound to cellular DNA and albumin as adducts, and nonsmokers experimentally exposed to SHS excrete NNAL, a carcinogenic nitrosamine found in tobacco smoke.

PREVALENCE OF TOBACCO USE [58]

The total no of tobacco users in the world has been estimated at 1.2 billion, which is expected to rise to 1.6 billion during 2020s.

Tobacco is used for smoking as well as in smokeless form in India. Smoking of tobacco is mainly in the form of beedi, followed by cigarette, hookah, chillum, chutta, etc., some common forms of smokeless tobacco include khaini, manipuri tobacco, mava, mishri,etc.,

Careful review of Indian studies concluded that beedi smoking is also associated with diseases caused by cigarette smoking and results in similar physiological changes. Despite their small size their tar and carbon monoxide deliveries are similar to those of manufactured cigarettes.

Tobacco use in India

According to national family health survey-2(1998 to 99),30% of population 15 years and above 47% men and 14.5% women-either smoked or chewed tobacco, which translates to almost 195 million people,154 million men and 41 million women in India.

Deaths due to tobacco use in India

Findings from 3 cohort studies (National Sample Survey Organization) suggested that at least 63,000 persons died in 1986 due to tobacco use. When the median risks as observed from these studies and prevalence of tobacco use, as found in the first nation-wide study, were applied to the 1996 population it showed that about 800,000 persons in India might have died due to the tobacco habit in India.

Magnitude of tobacco related diseases in India

Magnitude of three major tobacco related disease entities was estimated based on a careful review of Indian literature on risk estimates,

For development of these diseases in India and prevalence of tobacco use in India

Disease entity	Total no in India 1996	Cases due to tobacco use
Tobacco related cancer (incident cases)	209810	154,320
Coronary artery disease (prevalent cases)	15,700,000	4,200,000
Chronic obstructive lung diseases (prevalent cases)	14,000,000	3,700,000

PATHOPHYSIOLOGY OF SMOKING AND PATHOGENESIS OF ATHEROSCLEROSIS

The major forms of disease associated with smoking, i.e., coronary artery disease, stroke, peripheral artery disease are sequel of atherosclerosis. Atherosclerosis begins in childhood with accumulation of lipid in monocytes and smooth muscle cells of the arterial intima to form fatty streaks. Aortic fatty streaks, which appear shortly after birth, are present in almost all children shortly after 10 years of age and increase rapidly with age. Fatty streaks in the coronary arteries appear by about 15 and 20 years of age.

Fibrous plaques are elevated lesions with a core of lipid and necrotic debris with a cap of smooth muscle and connective tissue. These appear only rarely before 20 years of age but increase rapidly in prevalence and extent between20 and 30 years of age.

The fatty streaks progress to fibrous plaques at some anatomic sites by continued lipid deposition and proliferation of smooth muscle and connective tissue. However, those in other areas may remain innocuous fatty streaks or regress.

When fibrous plaques increase in size and undergo vascularisation, hemorrhage or ulceration and thrombosis, they lead directly to arterial occlusion and infarction of the affected organ, or in abdominal aorta, to weakening of the arterial media and formation of an aneurysm. Only at this stage does clinical disease become manifest.

Dyslipidemia- a risk factor for atherosclerotic disease

The importance of lipoproteins in the pathogenesis of atherosclerotic disease is well-known. The use of ultracentrifuge which allows separation of plasma lipoproteins provided important data on the relationship between LDL cholesterol, possible VLDL

cholesterol and coronary artery diseases. The role of HDL cholesterol as protective fraction also emerged.

LIPID EFFECTS OF SMOKING

Acute lipid effects of smoking [59]

Intravenous nicotine or nicotine inhaled from cigarette smoking raises plasma concentrations of Free Fatty Acids (FFAs) through enhanced lipolysis. FFA is mobilized from adipose tissue as a result of sympatho-adrenal stimulation. Increase in the delivery of FFAs to the heart results in increase in myocardial oxygen consumption through a metabolic stimulation independent of changes in mechanical activity of the heart. The higher incidence of myocardial infarction and ventricular arrhythmias in chronic smokers may be related to the elevated myocardial FFA concentration [20].

Chronic Lipid Effects

The atherogenic process is influenced by the levels of different plasma lipoproteins. Long term exposure to tobacco smoke enhances oxidation of LDL and decreases plasma HDL levels. The greater risk of atherosclerosis to people who are

exposed to tobacco smoking may result from the HDL lowering effects of tobacco smoke [59].

The free radicals entering the body are first trapped by serum aqueous and lipophilic antioxidants, which interact and provide greater protection against lipid peroxidation than any other antioxidant on its own. The total peroxyl radical trapping power of serum is calculated using sulfhydryl groups, vitamin E, uric acid and ascorbic acid. The end-products of lipid peroxidation in serum increases after passive smoking, which modify apo B in LDL, which leads to more rapid and unregulated uptake of LDL cholesterol by macrophages and as a result, formation of foam cells [67]. This modified LDL may also stimulate the release of free radicals, further modifying these lipoproteins.

The HDL class can be divided into subclasses HDL-2 and HDL-3. Current evidence indicates that the anti-atherogenic effect of HDL is related to the HDL-2 subclass. Smoking reduces both subclasses.

Smoking and hemostatic functions

Some of the detrimental effects of tobacco smoke on hemostatic function are increased leukocyte, platelet aggregation

and adhesiveness. Smokers have been found to have higher levels of plasma fibrinogen and there is a direct relationship between plasma fibrinogen levels and incidence of heart disease and stroke in population. Studies have also found that the levels of a metabolite of thromboxane A2 are elevated in smokers. In these people, the cardiovascular system is extremely sensitive to the effects of SHS than an active smoker.

NATIONAL CHOLESTEROL EDUCATION PROGRAM (NCEP) [61]

Adult treatment panel III

The adult treatment plan of the National Cholesterol Education Program (NCEP) released its recommendations concerning the diagnosis and management of hyperlipidemia in adults.

The risk factors recognized by ATPIII are as follows:

Major risk factors (Exclusive of LDL cholesterol that modify LDL goals)

- Cigarette smoking
- Hypertension

- Low HDL cholesterol
- Diabetes mellitus
- Family history of premature coronary artery disease
- Age (men 45 years; women 55 years)
- Lifestyle risk factors
- Obesity (BMI 30 kg/m2)
- Physical inactivity
- Atherogenic diet

Emerging risk factors

- Lipoprotein (a)
- Homocysteine
- Prothrombotic factors
- Pro-inflammatory factors
- Impaired fasting glucose
- Sub clinical atherogenesis

Note: HDL cholesterol 60mg/dl counts as a negative risk factor; its presence removes one risk factor from the total count.

Reference values

The recommendations in patients more than 20 years

Total cholesterol

Desirable : < 200 mg/dl

Borderline high : 200-239 mg/dl

High-risk : ≥240 mg/dl

LDL cholesterol-primary target of therapy

Optimal : <100 mg/dl

Near/above optimal : 100-129mg/dl

Borderline high : 130-159mg/dl

High : 160-189mg/dl

Very high : ≥ 190 mg/dl

HDL cholesterol

Low : <40mg/dl

High : ≥ 60mg/dl

Triglycerides

Desirable : <150 mg/dl

Borderline : 150-199 mg/dl

High risk : 200-499 mg/dl

Very High : ≥500mg/dl

Screening is recommended in all adults more than 20 years and should include a fasting lipid profile repeated every 5 years.

MANAGEMENT OF HYPERLIPIDEMIA [61]

The quantitative estimate of risk places individuals in one of the following treatment strata:

Risk category	LDL goal(mg/dl)	LDL level at which to initiate TLC(mg/dl)	LDL level at which to consider drug therapy
CHD or CHD risk equivalents (10 year risk >20%)	<100	100	130mg/dl
2+ risk factors (10 year risk 20%)	<130	130	10 year risk 10-20%:130mg/dl 10 year risk <10%:160mg/dl
0-1 risk factor	<160	160	190mg/dl

Note: ***CHD equivalents***

Symptomatic carotid artery disease Abdominal aortic aneurysm

Diabetes mellitus Peripheral arterial disease

TLC (Therapeutic Lifestyle Changes) diet

Saturated fat, 7% of calories, cholesterol-200 mg/day

Consider increasing viscous (soluble) fiber 10-25g/day), plant stanol/sterols (2g/day) as therapeutic options to enhance LDL lowering.

Weight management

Increased physical activity

Drug therapy

- Consider drug therapy simultaneously with TLC for CHD and CHD equivalents.
- Consider adding drug to TLC after three months for other risk categories.

Drugs affecting lipoprotein metabolism:

HMG CoA reductase inhibitors

e.g., Simvastatin, Pravastatin, Atorvastatin

Bile acid sequestrant resins

e.g., cholestyramine, cholestipol

Nicotinic acid and its derivatives

Fibrates

e.g., benzafibrate, fenofibrate

BENEFITS OF SMOKING CESSATION

Cessation of smoking consumption constitutes the single most important intervention in atherosclerotic disorders. Smoking cassation alone reduces the risk of a first heart attack by nearly 65 percent. Trials of nicotine replacement therapy using either transdermal nicotine or nicotine chewing gum have both proven to greatly increase abstention rates after cessation. Nicotine replacement during early abstinence helps to relieve symptoms of withdrawal and increase quit rates. Although the nicotine used as a medication may be addictive, it appears to be very minimal.

Although the elevated cardiovascular risk associated with smoking decreases significantly after smoking cessation, the risk of cancer of the lungs, pancreas and stomach persist for more than a decade as do the risks of developing chronic obstructive pulmonary disease. Thus primary prevention remains the most important population based component of any smoking reduction strategy [24].

MAJOR EFFORTS FOR TOBACCO CONTROL IN INDIA

Recognizing the health effects of tobacco, the Government of India promulgated The Cigarette (Regulation of Production, Supply and Distribution) Act 1975. Under the act, all packages and advertisements are to carry a statutory warning "Cigarette smoking is injurious to Health" [58].

The parliament committee on sub-ordinate legislation in its 22nd report December 1995) made wide ranging observations, including strong and rotatory warning in regional languages on tobacco products, prohibition of smoking in public places like hospitals, dispensaries, educational institutions, conference rooms, domestic air flights, trains, buses, initiation of measures for awareness on tobacco through health infrastructure, educational institutions and mass media and initiation of efforts for persuasion of farmers to switch over to alternate crops. These cabinet guidelines were reiterated in 1998.

Multi-sectoral approach for tobacco control

The problem of tobacco in India is complex in view of sectors like health, agriculture, finance, mass-media, labor,

education, industry, welfare, etc., unorganized nature of work for many tobacco products, dependence of a large number of people on tobacco production and processing and need for action by many agencies.

In July 1991, a national conference on tobacco and health was held. It recognized tobacco as a major public heath hazard and noted that consumption of tobacco is not compatible with the goal of 'Health for All'. It also recognized that an integrated educational, legislative and agro economic strategy with an operational framework. Political, administrative, financial and report support structure is needed. The conference recommended establishment of a National Tobacco Control commission to plan, co-ordinate and monitor tobacco control activities.

The Indian Council of Medical research (ICMR) conducted operational research product for involving schools and community volunteers in anti-tobacco education. The ICMR's collaborative project with All India Radio also showed the mechanism of inter-sectoral collaboration is cost-effective education through radio.

Community education on tobacco

The eradication of tobacco habits would require concerted action resulting in a social change. Anti-tobacco education needs to be targeted at decision-makers, professionals and the general public, especially the youth. No Tobacco Day (31st May) has been a regular feature since 1988, which generally comprises of educational advertisements in newspapers along with programs and workshops on tobacco. Tobacco has been included as a topic in books brought out by the NCERT.

Coverage of entire country for anti-tobacco education is a formidable job and cannot be achieved without active support from non-governmental organizations and mass media. They however, need support from the health departments for availability of reliable and impartial information on the subject. Support would be needed also from other related sectors like education, economics, agriculture, welfare, etc,

CONTROL OF ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

Exposure to SHS takes place in many different microenvironments. The contributions of different

microenvironments also depend upon age, sex, and other socio-demographic factors. For children, the home is a dominant locus of exposure, while for adults the workplace and social environments may be significant loci. Distinct control strategies are directed at different environments. Public places, workplaces, and transportation environments are subject to regulation while the home is not. Education is needed to reduce passive smoking in the home environment.

In general, the concentration of SHS in a particular indoor environment depends upon the intensity of smoking (the strength of the source), the rate of exchange of air in the space with outdoor air or other "clean" air, and the presence and effectiveness of any air cleaning devices. For public and commercial buildings, ventilation requirements are set by code. Air cleaning is also not sufficient if smoking is allowed [63].

Public places and transportation environments — Since the mid-1980s, there has been increasing regulation at the local and state level to restrict or eliminate smoking in public places. Model policies and regulations have been developed and made available by a number of organizations, including the American Lung Association. Exposure can be completely eliminated by

banning smoking indoors. Simply segregating smokers and nonsmokers within the same airspace may reduce the exposure of nonsmokers to SHS but does not eliminate it [7].

Workplaces —Model workplace smoking policies may involve either an outright ban or restriction of smoking to designated areas; some companies ban smoking altogether on their properties. In implementing a workplace smoking policy, smokers should be provided with opportunities to participate in smoking cessation programs and informed communication is needed for all employees. The efficacy of workplace smoking policies has been documented in studies involving measurement of airborne nicotine and also of the biomarker cotinine. One study demonstrated that policies that ban smoking in the workplace are the most effective and generally lower all nicotine concentrations.

The home environment — the air quality of homes is not subject to direct regulation, and effective educational strategies are needed to reduce exposures in homes through such steps as encouraging smokers to go outside the home or to restrict smoking to a particular room or away from children. Neither air cleaning nor increasing ventilation is a sufficient control measure. Typically in homes, indoor air exchanges with outdoor air through

natural diffusion and the air exchange rate cannot be substantially increased, even by opening windows and doors to the extent possible.

Air cleaners — A variety of devices are available for air cleaning, ranging from complex and expensive systems that are incorporated into the HVAC systems of buildings to simple tabletop devices and "smokeless ashtrays," that are purported to control SHS exposure at its source. In general, health care providers should advise against purchase of an air cleaning device as a strategy for controlling SHS exposure.

THE ROLE OF THE HEALTH CARE PROVIDER

Most smokers are aware of tobacco health risks and have a general knowledge of the risks of secondhand smoke exposure. However, they may not know about specific risks that might affect their decision to expose children or other family members to ETS. Smokers may not know that secondhand smoke exposure can cause lung cancer and cardiovascular disease in their spouses or contribute to asthma and otitis media in their children.

Smokers may be more amenable to change at key points in their lives or the lives of their children- pregnancy, birth of a child, early childhood (e.g., child begins to imitate smoking

behavior or asks about smoking), acute illness of the child that is related to smoking (e.g., otitis media), exacerbations of asthma. Healthcare providers should use these events as "teachable moments". Providing parents who smoke with guidance in cessation or referral to cessation programs is considered part of family-centered care. Families should be encouraged to actively control tobacco exposure as a way to role model a smoke-free attitude for the child.

Approaches for smoking cessation counseling are based upon the National Institutes of Health (NIH) and U.S. Public Health Service six-component clinical practice guidelines:

Anticipate - provide age-appropriate education to parents and children

Ask - ask about ETS exposure and parental tobacco use at every visit

Assess - assess if the smoker is ready to quit

Advise - advise parents and youth who smoke to quit

Assist - provide information and referrals for those who are ready to quit

Arrange follow-up - provide encouragement and help with relapse prevention.

In addition to encouraging abstinence, the physician should be familiar with counseling and therapeutic programs, strategies and intervention that promote cessation. Physicians should also participate in the public debate not only individually but also through medical societies and organizations.

RESPONDING TO SPECIFIC CLINICAL QUESTIONS AROUND SECONDHAND SMOKE

A number of situations that may arise in clinical practice and suggested approaches are given below. There are now extensive resources available on the health effects of passive smoking that can be used by physicians working in the public health domain.

Pregnancy —The majority of women stop smoking while pregnant, but a substantial percentage of smoking women (about 30 percent) continue to smoke; they need to be advised as to the adverse effects of their smoking for the fetus (including effects on growth and development) and for the continued adverse effects of their smoking after birth. The parents need to know that the father's smoking may be harmful to the unborn child as well.

Children at risk for asthma — Exposure to tobacco smoke should be avoided for the child at risk for asthma on the basis of a family history of asthma and allergies. During the first weeks after birth, infants whose mothers smoke have increased airway responsiveness, perhaps a precursor of asthma, and SHS exposure, particularly from maternal smoking, is a risk factor for asthma [13].

Children with asthma — SHS exposure, particularly from parental smoking, is associated with increased symptoms, medication usage, and utilization of health services for asthma [64]. Guidelines for the management of asthma call for the elimination of SHS exposure in the home for children with asthma.

Lung cancer and heart disease risk for adults — the causal conclusion with regard to lung cancer meant that nonsmokers should divorce smoking spouses. While divorce is a potential strategy to control exposure to SHS in the home, there are alternatives.

Workplace exposures — health care providers can reasonably postulate that the exposure may be harmful. If asked

about workplace risks from these exposures, health care providers can reasonably reply that the exposure would increase risk to some extent and that it could be reduced or eliminated with a workplace smoking policy.

Sick-building syndrome — Sick-building syndrome can be a complex clinical problem, requiring a clinical diagnosis in an individual to be based in a population context. Passive smoking should be considered as one of the factors that can contribute to the occurrence of sick-building syndrome.

PREPARING TO QUIT

Once you decide to quit smoking, the first step should be to set a quit date. Gradual reduction in smoking is occasionally successful as a means of quitting, but generally quitting "cold turkey" is more successful. Some people choose to switch to a brand of cigarettes that is lower in tar and nicotine as a prelude to quitting, but frequently this causes one to inhale more often or more deeply, counteracting the effect one tries to achieve.

BEHAVIORAL METHODS OF QUITTING

Behavioral approaches to smoking cessation can be done on your own or in individual or group sessions.

Problem solving/skills training — as mentioned, when preparing to quit smoking one needs to identify situations or activities that may increase his risk of smoking or relapse. Once one has identified his "danger situations," he needs to explore coping skills. Strategies to enhance coping may include making lifestyle changes to reduce stress and improve quality of life, such as starting an exercise program and learning relaxation techniques.

Social support — Social support can be very helpful in quitting smoking and staying off cigarettes. Social support systems may include family and friends, our health care provider, a counselor, a telephone hotline, and support groups.

Group counseling — Group programs are offered by a number of commercial and voluntary health organizations. They typically include lectures, group interactions, exercises on self-recognition of your habit, some form of a tapering method leading to a "quit day," development of coping skills, and suggestions for relapse prevention.

Hypnosis and acupuncture — Hypnosis and acupuncture are commercially popular stop-smoking methods.

PHARMACOLOGIC METHODS OF QUITTING

Nicotine replacement therapy and bupropion are the most commonly used medications for smoking cessation.

Nicotine replacement therapy — in the absence of nicotine, a smoker not only loses the good feelings nicotine induces but may also develop withdrawal symptoms. These include: depressed mood, insomnia, irritability, frustration or anger, anxiety, difficulty concentrating, restlessness, nicotine craving, decreased heart rate.

Nicotine replacement therapy is designed to reduce the intensity of these symptoms while a smoker deals with the behavioral aspects of smoking cessation. It will not prevent the symptoms completely. Nicotine replacement therapy appears to be acceptably safe, even in people with known cardiovascular disease. Many smokers are able to quit without using nicotine replacement therapy, although it is available to virtually anyone.

NICOTINE PREPARATIONS

Individual smokers may find one form particularly effective. Combinations of these therapies may be helpful in some people [2].

Nicotine polacrilex (gum) — Nicotine gum contains nicotine bound to a polacrilex resin that allows release of the nicotine with chewing. For most people who smoke one pack per day or more, use of 2 mg nicotine gum on an as-needed basis produces blood levels of nicotine less than 40 percent of those associated with usual smoking. As a result, the intensity of discomfort is often reduced. In addition, since peak blood levels of nicotine are much lower with gum than with a cigarette, both the feelings of well being and the addiction potential of nicotine gum are less than those of cigarettes. Nevertheless, many smokers have become chronic gum users. Treatment is generally recommended for three to six months.

There are disadvantages of using nicotine gum. Some people have a low oral pH that reduces the absorption of nicotine. Nicotine polacrilix lozenges are also available. They slowly release nicotine into the saliva in the mouth. The nicotine works similarly to the gum, as it must be absorbed in the mouth and not swallowed. Because the lozenges do not require chewing, they may be easier for some people to use.

Transdermal nicotine systems (patch) — Transdermal nicotine delivers nicotine to the blood through a

patch placed on the skin. The highest formulation (21 to 22 mg/patch) delivers nicotine at a rate that sustains a blood level about half as high as that found in a thirty cigarettes per day smoker. The combination of intensive behavioral programs and nicotine patches has produced very favorable smoking cessation rates. The patch may be more effective than the gum when an intensive behavioral program is not used. Treatment with nicotine patches is generally recommended at "full dose" for four to six weeks. The slow rise in blood nicotine levels with patches is believed to be associated with a low risk of addiction.

Nicotine nasal spray — compared to the patch and gum, the nasal spray produces a relatively rapid rise in nicotine concentration in the blood that more closely mimics changes seen with smoking. While studies have clearly demonstrated the safety and effectiveness of the nicotine nasal spray, it has a greater chance of prolonging nicotine dependence than the other therapies.

Nicotine inhaler — the nicotine inhaler has proved to be a safe and effective smoking cessation method. Because most of the nicotine is deposited in the mouth and delivered to the body through the mucous membrane in the mouth, nicotine absorption is relatively slow.

Bupropion — Bupropion is an antidepressant that has also been used to help a patient stop smoking. Bupropion may be more effective than nicotine replacement therapy, and combining the two may be even more effective. It is generally well tolerated, but it may cause dry mouth and insomnia.

RELAPSE

Smoking is now regarded as a relapsing disorder. Each quit should be regarded a victory, and the longer it lasts, the better. Relapse, however, may occur. Most relapses occur in the first week after quitting, when withdrawal symptoms are strongest. One should mobilize all his support resources (e.g., family, friends) during this critical time.

MATERIALS AND METHODS

The present study was conducted at the Institute of Internal Medicine, Govt. General Hospital, Chennai, between August 2007 and August 2007. One hundred married women in the age group of 20 to 45 years who had never smoked were selected among the patient attendants and volunteers attending the Master Health Clinic for routine health checkups. 50 subjects were non-smokers with self-reported exposure to ETS at home from the husband (case) and 50 subjects were non-smokers with a non-smoking partner (controls).

A standardized questionnaire was designed to collect information regarding demographic characteristics (age, residential are and occupation), history of hypertension, hyperlipidemia, diabetes mellitus, history of any drug intake and tobacco-chewing habits. Smoking and smoking habits of their husbands and other family members were noted in detail. Any exposure at workplace to ET was also noted. Subjects who use any form of tobacco were excluded from the study.

Physical examinations including anthropometric (wt/ht2) measurements were performed. Blood pressure was measured in

all subjects. Routine investigations like fasting blood sugar, serum creatinine, urea was done in all cases. Electrocardiogram was done in all cases.

Study group

Cases and controls were defined

(A) Cases (passive smokers)

Inclusion Criteria

Healthy married, non-smoking women with smoking husband with self-reported exposure to passive smoke at home for at least 1hr/day for consecutive 3 years or more with or without exposure to ETS at workplace.

Cases were also sub-grouped into two groups according to the duration of exposure

1. Exposed to ETS for 3 to 10 years.

2. Exposed to ETS for ≥ 11 years.

Exclusion Criteria

1. Tobacco chewing habits
2. Subjects on any regular medications
3. Oral contraceptive pill intake

4. Family history of premature deaths
5. Pregnancy and lactation
6. Body-mass index ≥30
7. Diabetes mellitus, hypertension, IHD, hypothyroidism, nephritic syndrome, chronic liver diseases
8. Alcohol intake
9. Post-menopausal women
10. Malignancies, steroid therapy

(B) Control (Non-smokers)

Healthy married women who were not exposed to passive smoke at home or at workplace. The dietary habits were almost the same among cases and controls. Same set of exclusion criteria was applied for controls also.

Study design

Since only females were included, it was a gender matched case-control study.

Blood collection, storage & stability

Venous blood samples were taken after 12 hours of overnight fasting from ante-cubital vein of horizontally resting

arm with 21 gauge needle; samples were transported to the lab within 1 hour. Serum is preferred. Heparinised or EDTA plasma can also be used. Serum or plasma samples should be used as soon as possible on the same day.

Blood analysis

Fasting blood sugar serum urea and serum creatinine were estimated. Fasting serum concentration of total cholesterol was estimated by enzymatic method, HDL cholesterol by phosphotungstate method and triglycerides by enzymatic calorimetric method by using commercial kits (from Bayer Diagnostics India Ltd) and the Express Plus auto-analyzer. Uric acid estimation was done by the uricase method.

Estimation of cholesterol (Enzymatic method)

Principle

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{cholesterol esterase}} \text{Cholesterol} + H_2O_2$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{cholesterol oxidase}} \text{Chlesterol} + H_2O_2$$

$$2H_2O_2 + \text{phenol} + \text{4-Aminoantipyrene} \xrightarrow{\text{peroxidase}} \text{Red quinine} + 4H_2O_2$$

One ml of reconstituted reagent containing enzymes/chromogen and buffer (pipes buffer, Phenol, Sodium cholate) and 10 microlitre of the sample serum were mixed in a test tube, incubated at 37°Celsius for 5 minutes and read at 500 nm. The concentration of cholesterol in the sample is directly proportional to the intensity of the red complex (red quinine)

Estimation of HDL cholesterol (Phosphotungstate method) [26]

Principle

Chyomicrons, VLDL and LDL fractions in serum or plasma are separate from HDL by precipitating with phosphotungstic acid and magnesium chloride. After centrifugation the cholesterol in the HDL fraction, which remains in the supernatant is assayed by enzymatic method using the cholesterol esterase, cholesterol oxidase, peroxidase and the chromogen 4-aminoantipyrene and buffer

Estimation of triglycerides (Enzymatic Method)

Principle

$$\text{Triglycerides} + H_2O \xrightarrow{\text{lipoprotein lipase}} \text{Glycerol} + \text{Fatty Acid}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{glycerol kinase}} \text{Glycerol-3-phosphate} + \text{ADP}$$

$$\text{Glycerol-3-phosphate} + O_2 \xrightarrow{\text{GPO}} \text{Dehydroxyacetone phosphate} + H_2O_2$$

$$2H_2O_2 + \text{4-aminoantipyrene} + \text{ADPS} \xrightarrow{\text{peroxidase}} \text{Red quinone} + 4H_2O$$

GPO glycerol-3-phosphate oxidease

ADPS N-ethyl-n-Sulfopropyl-n-anisidine

The intensity of purple colored complex formed during the reaction is directly proportional to the triglycerides concentration in the sample and is measured at 546nm.

LDL cholesterol was calculated according to the Friedwald equation.

LDL=TC─ (HDL + Triglycerides/5)

VLDL is calculated by the formula

VLDL=TC─ (HDL+LDL)

Uric Acid (Enzymatic method)

This reagent kit is used for in vitro quantitative determination of uric acid in serum, plasma or urine.

Principle:

Uric acid is converted by uricase into allantoin and hydrogen peroxide in the presence of peroxidase (POD) oxidizes the chromogen into a red colored compound which is read at 500nm(492-550). The final color of the reaction is stable for fifteen minutes.

$$\text{Uric acid} + 2H_2O + O_2 \xrightarrow{\text{uricase}} \text{Allantoin} + CO_2 + H_2O$$

$$H_2O_2 + \text{4- Aminoantipyrine} + \text{DHBS} \xrightarrow{\text{POD}} \text{Red quinone} + H_2O + HCl$$

DHBS 3, 5- Dichloro-2-Hydroxybenzene Sulfonic Acid

OBSERVATION AND RESULTS

The lipid profile obtained from 100 subjects, among whom 50 were passive smokers (cases) and 50 non-smokers (controls) was analyzed.

Distribution of cases and controls in relation to age-group

Table 1: Distribution of cases and controls in relation to age

Age(years)	Cases(passive smokers) No(%) n=50	Controls(Non-smokers) NO.(%) n=50
20-25	4(8)	6(12)
26-30	23(46)	20(40)
31-35	15(30)	15(30)
36-40	8(16)	5(10)
41-45	0(0)	4(8)

Total Cholesterol

The mean Total Cholesterol value in subjects not exposed to ETS (controls) was 166.08 ± 26.45 mg/dl, whereas the total cholesterol in women exposed to ETS was 181.64 ± 30.42 mg/dl (p=0.0104,statistically significant).

Table 2: Total cholesterol in non-smokers and passive smokers

	Numbers(n)	Mean total cholesterol (mg/dl)	SD
Non-smokers(control)	50	166.08	26.45
Passive smokers(cases)	50	181.64	30.42

P=0.0104

LDL Cholesterol levels

The mean LDL Cholesterol in non-smokers was 96.44 ±28.42 mg/dl whereas the LDL cholesterol in passive smokers was 113.27±34.54 (p=0.0089, statistically significant).

Table 3: LDL cholesterol in non-smokers and passive smokers

	Numbers(n)	Mean LDL cholesterol (mg/dl)	SD
Non-smokers(control)	50	96.44	28.42
Passive smokers(cases)	50	113.27	34.54

p=0.0089, statistically significant

HDL cholesterol levels The mean HDL cholesterol in non-smokers was 46 ± 9.3 whereas in passive smokers the mean HDL-C was 43.98 ± 7.32(p=0.2248, statistically not significant).

Table 4: HDL cholesterol in non-smokers and passive smokers

	Numbers(n)	Mean HDL cholesterol (mg/dl)	SD
Non-smokers(control)	50	46	9.3
Passive smokers(cases)	50	43.98	7.2

(p=0.2248, statistically not significant).

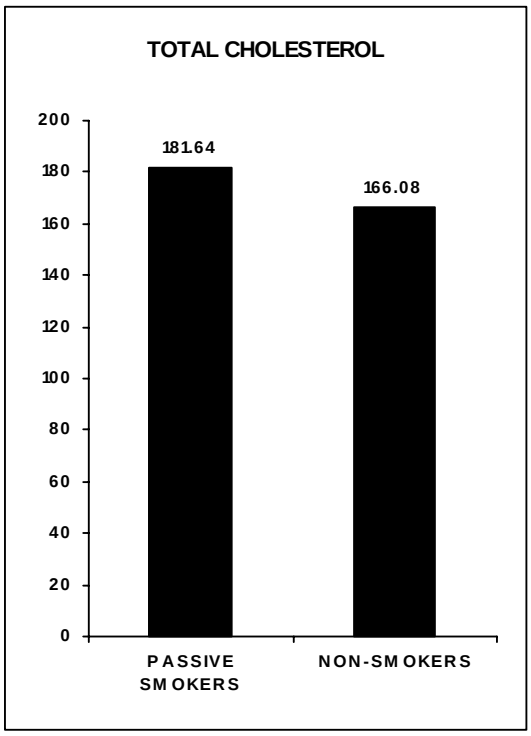
Triglycerides

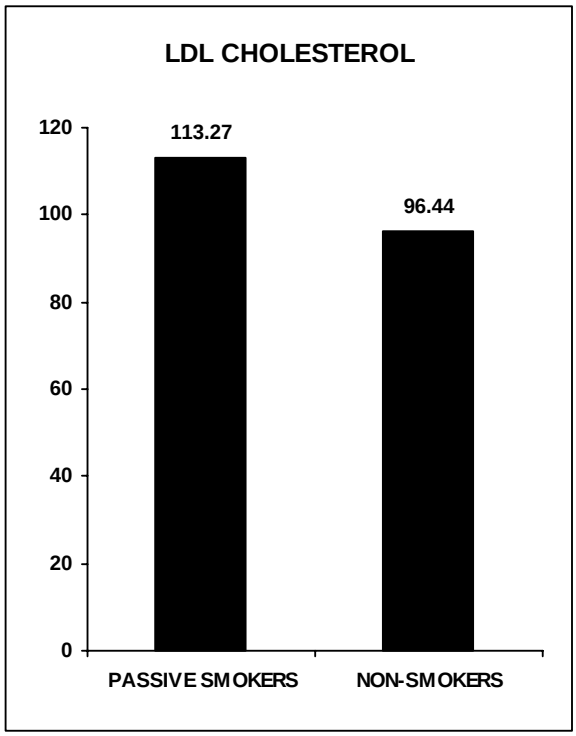
The mean triglycerides in non-smoking women was 119.74± 29.56 whereas the mean triglycerides in passive smokers was 126.1±28.54 (p=0.275, statistically not significant).

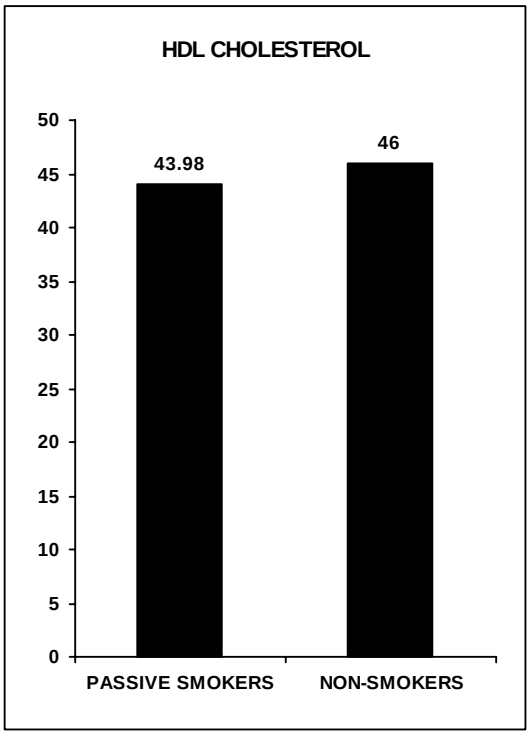
Table 5: Triglycerides in non-smokers and passive smokers

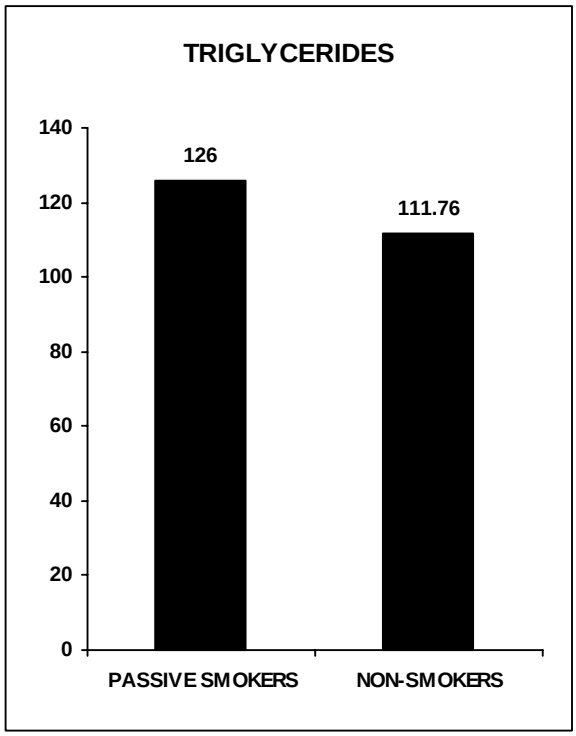
	Numbers(n)	Mean TGL (mg/dl)	SD
Non-smokers(control)	50	119.76	29.56
Passive smokers(cases)	50	126.1	28.54

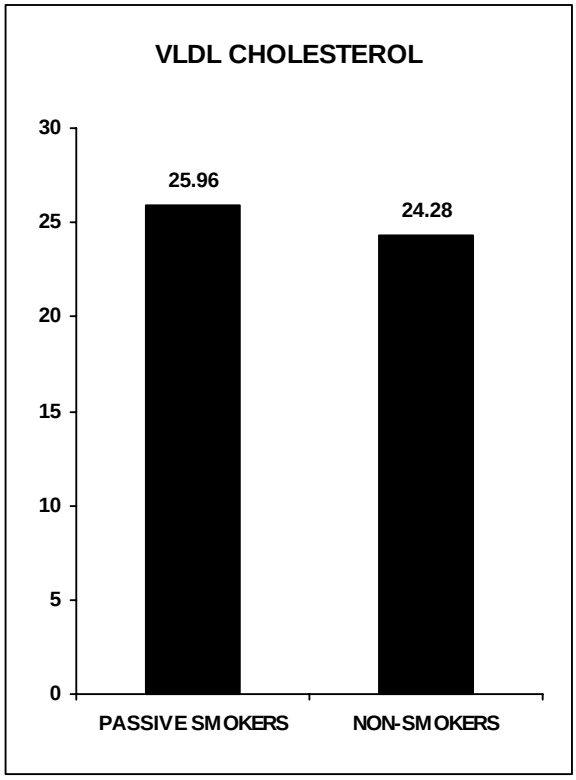
(p=0.275, statistically not significant).

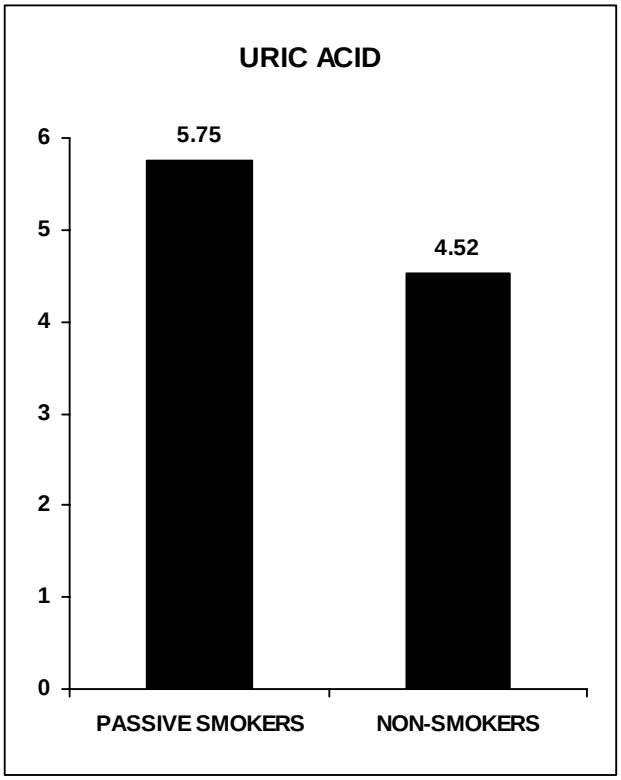

TOTAL CHOLESTEROL
200
180
160
140
120
100
80
60
40
20
0
181.64
166.08
PASSIVE SMOKERS
NON-SMOKERS


LDL CHOLESTEROL
120
100
80
60
40
20
0
113.27
96.44
PASSIVE SMOKERS
NON-SMOKERS


HDL CHOLESTEROL
50
45
40
35
30
25
20
15
10
5
0
43.98
46
PASSIVE SMOKERS
NON-SMOKERS


TRIGLYCERIDES
140
120
100
80
60
40
20
0
126
111.76
PASSIVE SMOKERS
NON-SMOKERS


VLDL CHOLESTEROL
30
25
20
15
10
5
0
25.96
24.28
PASSIVE SMOKERS
NON-SMOKERS


URIC ACID
6
5
4
3
2
1
0
5.75
4.52
PASSIVE SMOKERS
NON-SMOKERS

VLDL Cholesterol levels

The mean level of VLDL in non-smoking women was 24.28±5.96 mg/dl whereas VLDL in those exposed to ETS was 25.96±5.7, (p=0.235, statistically not significant).

Table 6: VLDL Cholesterol in non-smokers and passive smokers

	Numbers(n)	**Mean VLDL(mg/dl)**	**SD**
Non-smokers(control)	50	24.28	5.96
Passive smokers(cases)	50	25.96	5.7

p=0.235, statistically not significant

Serum uric acid

The mean level of serum uric acid in non-smoking women was 4.52mg/dl whereas uric acid in those exposed to ETS was 5.75mg/dl, (p=0.002, statistically significant).

Table 7: Serum uric acid in non-smokers and passive smokers

	Numbers(n)	Mean serum uric acid(mg/dl)	SD
Non-smokers(control)	50	4.52	1.47
Passive smokers(cases)	50	5.75	1.6

p=0.002, statistically significant

Table 8: The lipid profile in women exposed to ETS with relation to duration of exposure

Lipids	Exposed to ETS 3-10 years n=27	Exposed to ETS≥11 years n=23	P value
Mean Cholesterol(mg/dl)±SD	176.96±36.52	187.17±23.43	0.2546
Mean LDL mg/dl ±SD	108.19±39.10	119.25±27.7	0.2617
Mean HDL mg/dl ±SD	45.70±7.24	41.96±6.79	0.064
Mean triglycerides mg/dl ±SD	120.11±29.7	133.13±25.78	0.107
Mean VLDL mg/dl	24.70±5.94	26.43±5.1	0.09

DISCUSSION

Both the cases (exposed to ETS) and controls (not exposed to ETS) were comparable in terms of age, weight, body mass index and dietary habits. All the subjects were pre-menopausal women and all were housewives without exposure to ETS at workplace.

The mean serum Total Cholesterol value among the non-smokers was 166.08 ± 26.45 while in the passive smokers it was 181.64 ± 30.42. The value was significantly higher in passive smokers. MP HOLAY et al [69] (2004) have demonstrated that mean cholesterol level is higher in passive smokers (169 ± 27.6 mg/dl) than in non-smokers (151.6 ± 13 mg/dl). In the study conducted by Feldman et al (1991) [66] in adolescents, it was found that the ratio of total cholesterol to HDL cholesterol was 8.9% greater in passive smokers than in non-smokers. These results suggest that passive smoking like active smoking leads to alteration in lipid profile predictive of an increased risk of atherosclerosis.

The mean LDL cholesterol level was 96.44 ± 28.42mg/dl in non-smoking women while the LDL Cholesterol in passive smokers was 113.27± 34.54 mg/d [67]. This is in accordance with the study conducted by VALKONEN M et al who observed that mean LDL cholesterol in passive smokers was 110.33±27.6mg/dl and in non-smokers was 93.54±26.23 mg/dl. MP HOLAY et al (2004) [69] observed high mean LDL levels (106±20.4mg/dl) in passive smokers than in non-smokers (97.26±15.5mg/dl).

Acute exposure of a non-smoking subject to passive smoking resulted in deterioration of serum oxidant defense, accelerated lipid peroxidation and accumulation of LDL cholesterol in human macrophages. These events were observed even after a very short period (30 minutes) of passive smoking. Indeed it has previously been suggested that the cardiovascular system is extremely sensitive to the chemicals in environmental tobacco smoke. Further the oxidative stress induced by second hand smoke may have more prominent effects on a non-smoking subject than an active smoker whose cardiovascular system has adapted to cigarette smoke [67, 68].

The mean HDL cholesterol in non-smokers was higher (46±9.3 mg/dl) than in passive smoking women (43.98±7.2

mg/dl). This is accordance with studies by ELLIS J. NEUFELD et al (1997) [70] who found out that the mean HDL Cholesterol levels in children from households with cigarette smokers were lower (38.7±1.2mg/dl) than in controls (43.6±mg/dl). In the study conducted by MP HOLAY et al (2004) the HDL Cholesterol level was 51.48±6mg/dl in passive smokers and 55.24±6.7mg/dl in non-smokers [69].

The mean triglycerides in non-smoking women were 119.76±29.56 mg/dl whereas in passive smoking women it was 126.1±28.46mg/dl. This is similar to that observed by MP HOLAY et al (2004) [69] who found that mean triglycerides in passive smokers was 125.76±19.2 mg/dl and in non-smokers was 116.6±14.20mg/dl.

Mean VLDL Cholesterol is also higher in passive smokers (25.96±5.7mg/dl) than non-smokers (23.58±5.96mg/dl).

In the present study as shown, total cholesterol, LDL and VLDL increase with duration of exposure to ETS, though statistically not significant. There is considerable reduction in levels of HDL with increasing duration of exposure to ETS.

The mean serum uric acid in non-smokers was 4.52±1.47 mg/dl whereas in those exposed to ETS it was 5.75±mg/dl. Miia Valkonen and Timo Kuusi had shown similar results in their studies [67].

STUDY LIMITATIONS

1. The study is a hospital-based study and may not be representative of the general population.
2. There may be alternative pathways that may be responsible for cardiovascular complications in passive smoking.
3. This study does not elucidate the causal relationships between the risk factors and outcome.

SUMMARY

1. The mean serum Total Cholesterol value among the non-smokers was 166.08 ± 26.45 while in the passive smokers it was 181.64 ± 30.42. The value was significantly higher in passive smokers.

2. The mean LDL cholesterol level was 96.44 ± 28.42mg/dl in non-smoking women while the LDL Cholesterol in passive smokers was 113.27± 34.54 mg/d.

3. The mean HDL cholesterol in non-smokers was higher (46±9.3 mg/dl) than in passive smoking women (43.98±7.2 mg/dl).

4. The mean triglycerides in non-smoking women were 119.76±29.56 mg/dl whereas in passive smoking women it was 126.1±28.46mg/dl.

5. Mean VLDL Cholesterol is also higher in passive smokers (25.96±5.7mg/dl) than non-smokers (23.58±5.96mg/dl).

6. The mean serum uric acid in non-smokers was 4.52±1.47 mg/dl whereas in those exposed to ETS it was 5.75±mg/dl.

CONCLUSION

Passive smoking like active smoking is unfavorably associated with lipoprotein metabolism and it predisposes the individual to premature atherosclerosis and its consequences like myocardial infarction, stroke, etc., Passive smoking also increases serum uric acid and this may serve as a crude marker to the amount of oxidative stress imparted to the body by the process of passive smoking.

To conclude the present study provides further evidence that environmental tobacco smoke including smoke at workplace is likely to cause adverse health effects in non-smoking women. Despite the efforts to control smoking most women are still exposed to tobacco smoke at home and at work. They are also unaware of the hazards of passive smoking and are in a disadvantaged position to protect themselves from ETS. Urgent public health measures are required to reduce smoking in India so as not only to protect women and children from environmental tobacco smoke but also men from the hazards of active smoking.

SCOPE FOR FURTHER STUDIES

The exact role of the changes in the various lipid components in the pathogenesis of atheromatous changes is still to be elucidated. The SHS-induced alterations in the lipid profile have just been thrown light upon by this study. The various mechanisms of uptake of the altered lipids by the macrophages and their conversion to atherogenic foam cells is an interesting and a potentially fruitful field to work up on.

Ascorbic acid, sulfhydryl groups, uric acid, vitamin C and vitamin A have been postulated as the first line of defense against the added anti-oxidants added to any body system. Research work is possible on the various other less-studied and more specific components of the anti-oxidant system.

Pharmacology oriented studies regarding the combination therapies for smoking cessation is a possible arena of interest.

Physicians interested in preventive medicine shall take up a possible research on the environmental aspects of SHS control.

ANNEXURE

PROFORMA

CHANGES IN SERUM LIPID LEVELS, URIC ACID DUE TO PASSIVE SMOKING IN HEALTHY FEMALES

1. NAME
2. AGE
3. SEX
4. OP NO
5. RESIDENTIAL
6. OCCUPATION
7. MENSTRUAL HISTORY
8. HISTORY OF TREATMENT FOR HYPERTENSION
9. HYPERLIPIDEMIA
10. DRUG INTAKE
11. TOBACCO CHEWING HABITS
12. SMOKING HABITS OF HUSBAND/OTHER FAMILY MEMBERS
13. SMOKE EXPOSURE AT WORKPLACE
14. HISTORY OF ORAL CONTRACEPTIVE PILL INTAKE
15. FAMILY HISTORY OF PREMATURE DEATHS

16. PREGNANCY AND LACTATION

17. HISTORY OF TREATMENT FOR ISCHEMIC HEART DISEASE

18. HYPOTHYROIDISM

19. NEPHROTIC SYNDROME

20. CHRONIC LIVER DISEASE

21 ALCOHOL INTAKE

EVALUATION

1. HEIGHT

2. WEIGHT

3. BODY MASS INDEX

4. BLOOD PRESSURE

5. FASTING BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

6. FASTING LIPID PROFILE

7. SERUM URIC ACID

ABBREVIATIONS

1. ETS: Environmental tobacco smoke
2. IARC: International Agency for Research on Cancer
3. SHS: Secondhand smoke
4. SIDS: Sudden infant death syndrome
5. SMR: Standardized mortality ratio
6. CAD: Coronary artery disease
7. LDL: Low-density lipoprotein
8. HDL: High-density lipoprotein
9. VLDL: Very low density lipoprotein
10. TC: Total cholesterol
11. TGL: Triglycerides
12. CalEPA: California Environment Protection Agency
13. NCEP: National Cholesterol Education Program
14. ATP III: Adult treatment plan III
15. ICMR: Indian Council for Medical Research
16. EDTA: Ethylene diamine tetra-acetic acid

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MASTER CHART

CONTROL

S.No	Name	Age	Sex	Duration of exposure to ETS		Blood biochemistry (mg/dl)			Serum lipid (mg/dl)					Serum uric acid (mg/dl)
				At home	At work	Sugar (F)	Urea	Creatinine	Total cholesterol	LDL	HDL	TGL	VLDL	
1	Dhanalakshmi	25	F	nil	nil	92	22	0.8	171	110	41	94	20	2
2	Durga	30	F	nil	nil	98	26	1	122	68	43	71	15	3
3	Geetha	25	F	nil	nil	96	22	0.8	179	104	46	103	21	5.1
4	Jayanthi	28	F	nil	nil	94	24	0.8	213	149	41	122	25	6.2
5	Suseela	30	F`	nil	nil	97	22	0.8	199	132	38	104	21	6
6	Parvathi	32	F	nil	nil	101	23	1	184	127	38	102	21	3
7	Usha	33	F	nil	nil	96	22	1	148	82	41	133	21	5.2
8	Pushpa	26	F	nil	nil	98	24	0.8	147	83	45	102	21	5.5
9	Priya	42	F	nil	nil	90	20	0.8	167	102	45	106	22	7
10	Santhi	28	F	nil	nil	109	26	1.2	156	96	39	111	23	4.2
11	Kamala	25	F	nil	nil	116	24	0.8	166	94	46	135	28	5
12	Maragatham	29	F	nil	nil	110	26	0.8	116	63	36	88	19	4
13	Muniyammal	28	F	nil	nil	100	20	0.8	111	44	41	134	28	7
14	Sakunthala	29	F`	nil	nil	90	20	1.2	146	92	31	120	25	4
15	Samruthbegum	33	F	nil	nil	90	23	1.1	161	87	55	103	21	6.1
16	Vasantha	35	F	nil	nil	108	22	0.8	172	118	33	111	23	7
17	Rani	38	F	nil	nil	97	22	0.8	177	114	41	115	24	4.5
18	Kamatchi	35	F	nil	nil	72	20	0.8	194	140	31	123	25	1.9
19	Kala	29	F	nil	nil	76	16	0.8	201	140	39	119	24	2.8
20	Rukmani	38	F	nil	nil	78	20	0.8	158	98	43	91	19	4
21	Arogyamary	27	F	nil	nil	90	22	0.8	151	88	31	168	34	6.6
22	Gunasundari	42	F	nil	nil	100	20	1.1	147	98	31	96	20	3.9
23	Krishnaveni	35	F`	nil	nil	117	30	0.8	161	105	45	63	13	4.6

24	Bhuvaneswari	32	F	nil	nil	78	22	0.8	157	95	45	93	19	2.9
25	Sanmugapriya	42	F	nil	nil	90	20	1	172	107	41	125	26	3.7
26	Radha	33	F	nil	nil	80	20	0.8	243	154	45	102	21	5.1
27	Dhanaselvi	26	F	nil	nil	90	26	0.8	131	49	59	121	33	5.9
28	Gunaselvi	31	F	nil	nil	100	26	0.9	171	78	63	154	25	6
29	Prabhavathi	29	F	nil	nil	98	24	0.8	165	85	66	127	32	3
30	Sampornam	24	F`	nil	nil	96	22	0.8	165	85	56	127	32	5.2
31	Malathi	27	F	nil	nil	105	24	0.8	191	115	39	189	24	5.5
32	Sudha	26	F	nil	nil	100	23	0.8	176	90	55	161	39	3.1
33	Gandhamani	31	F	nil	nil	96	20	0.8	137	56	63	95	20	4.2
34	Valli	28	F	nil	nil	88	24	0.8	141	57	61	119	25	5
35	Ramani	25	F	nil	nil	86	22	0.9	216	139	41	190	39	4
36	Ponnammal	25	F	nil	nil	90	22	1	181	95	56	157	32	3.9
37	Manjula	29	F	nil	nil	104	22	1	201	128	35	196	40	4
38	Chinnammal	33	F	nil	nil	90	20	0.8	165	87	57	111	23	6.1
39	Sangeetha	26	F`	nil	nil	84	24	0.7	131	45	52	177	36	7
40	Isaimathi	42	F	nil	nil	80	22	0.9	103	37	49	92	19	4.5
41	Hemavathi	28	F	nil	nil	90	22	0.8	151	77	51	122	25	1.9
42	Biwi Jahn	23	F	nil	nil	90	20	0.8	148	76	56	87	18	2.8
43	Pushpalatha	27	F	nil	nil	98	26	0.7	147	74	39	145	30	4
44	Parvathi	38	F	nil	nil	106	26	0.9	204	137	51	93	19	5.8
45	Anitha	34	F	nil	nil	96	28	0.7	197	131	46	106	22	3.9
46	Kanagavalli	25	F	nil	nil	90	20	0.9	205	115	66	125	26	4.6
47	Aswini	26	F	nil	nil	100	22	1	176	108	45	121	25	2.9
48	Palaniammal	31	F`	nil	nil	96	20	1.1	166	35	55	138	28	7.7
49	Thilagavathi	28	F	nil	nil	86	20	1.2	153	87	45	109	23	2.1
50	Ramanammal	40	F	nil	nil	83	35	1.0	169	65	45	101	21	3

CASES

S.No	Name	Age	Sex	Duration of exposure to ETS		Blood biochemistry (mg/dl)			Serum lipid (mg/dl)					Serum uric acid (mg/dl)
				At home	At work	Sugar (F)	Urea	Creatinine	Total cholesterol	LDL	HDL	TGL	VLDL	
1	Chinnamani	29	F	10	nil	90	20	0.8	130	48	58	120	24	3.7
2	Chitra	35	F	15	nil	98	26	0.9	171	78	63	154	32	8.1
3	Mangalam	30	F	6	nil	106	36	0.8	223	150	35	196	40	5
4	Dhanakodi	28	F	5	nil	96	28	0.8	165	87	57	111	23	4
5	Anandhi	29	F`	6	nil	98	24	1	191	105	52	177	36	5.7
6	Kumari	27	F	5	nil	105	22	0.8	103	37	49	92	19	6.8
7	Santhakumari	26	F	5	nil	100	20	0.8	151	77	51	122	25	4
8	Jayammal	29	F	8	nil	100	20	0.8	151	77	51	122	25	5.9
9	Mahalakshmi	30	F	9	nil	88	22	1	121	42	59	105	22	8
10	Babyammal	32	F	11	nil	86	22	1	155	93	37	129	27	9.1
11	Padma	28	F	9	nil	90	22	0.8	276	210	43	123	25	3.7
12	Pattu	32	F	12	nil	104	24	0.8	186	127	41	94	20	8.1
13	Bhagyam	40	F	18	nil	80	22	1.2	191	118	47	137	28	3
14	Adhilakshmi	35	F`	14	nil	84	24	0.8	184	123	43	95	20	3.6
15	Seetha	23	F	3	nil	80	22	0.8	222	162	44	88	18	7.5
16	Rajeswari	30	F	10	nil	90	22	0.8	184	123	43	95	20	3.1
17	Maliga	27	F	7	nil	90	20	0.8	160	98	41	112	23	6.2
18	Nagammal	23	F	4	nil	98	26	0.9	157	88	47	65	14	3.9
19	Mangammal	40	F	20	nil	106	26	1	225	150	47	147	30	3
20	Saroja	35	F	14	nil	96	28	1	166	108	36	117	24	6.4
21	Ammu	30	F	9	nil	90	20	0.8	191	128	42	129	23	7
22	Saradha	36	F	15	nil	100	22	0.7	223	152	50	111	23	4.5

23	Pachaiyamal	33	F`	11	nil	96	20	0.9	204	129	48	141	29	4.6
24	Sowri	34	F	11	nil	87	20	0.8	177	116	40	113	23	7.3
25	Pavunammal	27	F	5	nil	90	20	0.8	186	120	41	131	27	8.8
26	Panchavarnam	28	F	15	nil	90	22	0.8	151	98	32	111	22	5
27	Tamilselvi	35	F	12	nil	90	20	0.8	130	25	48	141	29	4.4
28	Rebecca	28	F	5	nil	98	26	0.8	182	130	28	126	26	4.5
29	Savithri	38	F	15	nil	106	26	0.9	203	135	37	161	33	7
30	Govindammal	30	F`	9	nil	96	28	1	226	150	50	134	28	6
31	Deepa	33	F	13	nil	90	20	1	185	150	50	134	28	5.9
32	Bommi	37	F	17	nil	100	22	0.8	215	149	38	145	30	7.2
33	Periyathai	40	F	17	nil	96	20	0.7	171	105	42	127	26	6.8
34	Anjalai	32	F	11	nil	86	20	0.9	172	116	36	94	20	7.7
35	KhurshedBegum	40	F	18	nil	96	22	1	181	126	34	10	23	4.7
36	Indra	27	F	7	nil	90	20	0.8	187	116	46	133	27	8.1
37	Kalavathi	28	F	9	nil	98	26	0.8	157	83	41	169	35	4.1
38	SaithoonBeevi	30	F	9	nil	96	22	0.9	161	103	41	91	19	9
39	Sengammal	30	F`	9	nil	95	24	0.8	219	165	43	109	23	10
40	Yasodha	35	F	16	nil	96	22	0.8	193	121	37	179	37	5.7
41	Alamelu	35	F	15	nil	98	24	0.8	198	128	37	173	35	4
42	Rajasri	27	F	7	nil	90	20	0.7	138	63	53	117	24	6.2
43	Manimegalai	26	F	5	nil	108	26	0.9	181	127	35	102	21	3.1
44	Kamroonnisha	22	F	3	nil	96	24	0.8	168	105	41	117	24	4.1
45	Nithya	39	F	18	nil	110	26	0.8	200	131	41	147	30	6.8
46	Andal	33	F	10	nil	90	20	0.7	199	121	47	159	33	7.9
47	Dharani	28	F	7	nil	90	20	0.9	180	111	47	114	24	4.9
48	Panchavarnam	24	F`	3	nil	108	23	0.7	180	110	48	74	16	3
49	Tamilselvi	35	F	15	nil	97	22	0.9	199	144	43	167	34	4.3
50	Rebecca	35	F	14	nil	92	22	1	203	128	46	153	31	6.1